

## **POSTER ABSTRACT ADDENDUM**

### **P68a**

**MEASURING INCREASES IN MESOLIMBIC DOPAMINE AND RELATED SUBSTANCES IN RESPONSE TO SYSTEMIC NICOTINE USING IN VIVO ELECTROCHEMISTRY.** G.C. Parker and P.B.S. Clarke Dept. of Pharmacology, McGill University, Montreal, Canada H3G 1Y6.

Many of the psychopharmacological properties of tobacco smoking are believed to be mediated by central nicotinic receptors (nAChRs). The locomotor stimulant and reinforcing effects of nicotine in rats are blocked by a selective loss of mesolimbic dopamine innervating the ventral striatum. Using selective and non-selective electrochemical probes, we have monitored the release of DA alone and also the levels of DOPAC and ascorbate, in the nucleus accumbens following systemic administration of nicotine (0.4 mg/kg s.c.) in unanaesthetized freely-moving rats. The increase in current observed at the dopamine-selective probe was seen to be maximal within 20 min and returned to baseline after 60 min. In contrast, the increase in signal at the non-selective probe was maximal at 2 hr and returned to baseline after 5 hr. The superior frequency of sampling afforded by in vivo electrochemistry has clear advantages. In particular, it should enable us to characterize rapid neurochemical changes in response to nicotine when the drug is given in ways that more closely mimic the pharmacokinetics of smoking.

### **P91**

**CHRONIC NICOTINE-INDUCED COGNITIVE FACILITATION ARE CORRELATED WITH CHANGES IN THE NICOTINE BINDING SITES.** F.A. Abdulla, E.J. Bradbury, M.-R. Calaminici, S. Wonnacott, J.D. Stephenson, J.D. Sinden and J.A. Gray. Dept. of Pharmacology, University of Alberta, Canada, Depts. of Neuroscience and Psychology, Institute of Psychiatry, London, and Dept. of Biochemistry, University of Bath, Bath, U.K.

The present study investigated the role of nicotinic receptors in chronic nicotine-induced cognitive facilitation. (-)-Nicotine tartrate (2mg/kg) and mecamylamine (1.0 mg/kg) were administered to different groups of rats twice daily for 10 days. A third group received the same dose of nicotine for one day and the vehicle (saline) for 9 days. Beginning 24h after the final drug injection, the rats were compared to a vehicle control group on acquisition of a hidden platform in the Morris water maze over 20 trials with 30-min inter-trial interval. The rats were killed 48h after the last drug injection and their frontal cortex (FC), entorhinal cortex (EC), posterior cingulate cortex (PC), dorsal hippocampus (DH) and ventral hippocampus (VH) were rapidly dissected to be assayed for nicotinic binding sites. Chronic treatment with nicotine significantly increased the number of FC, EC and DH but not the PC or the VH binding sites and improved the rate of learning. Chronic treatment with mecamylamine significantly increased the number of FC but not the other regions nicotinic receptors and decreased the rate of learning. Nicotine given for one day increased the EC (but not other regions) binding sites and increased the rate of learning but failed to match the rats receiving nicotine for 10 days. There were significant correlations between the EC, DH and PC (but not FC or VH) nicotinic receptor numbers and the rate of learning during acquisition but not with performance at asymptote. The results may indicate that chronic nicotine increased cholinergic transmission which in turn induced learning facilitation. Supported by R.J. Reynolds Tobacco Co.

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